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# Determinants of environmental tobacco smoke in a population of Puerto Rican children

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**This study was designed to determine among various personal, socioeconomic, and environmental factors those which had the greatest influence on exposure to environmental tobacco smoke (ETS) in a population of children residing in a tropical environment and to compare these results with those obtained in the literature of tobacco exposed children in temperate climates. Urine specimens were collected from 606 healthy Puerto Rican children (2–12 years) living in an industrial area and analyzed for cotinine, a quantitative biomarker for exposure to ETS. Parents completed a questionnaire covering smoking habits and socioeconomic information. Seventy per cent of the children were reported to be exposed to ETS, 50% resulting from exposure to smoke from either or both parents. Major determinants to ETS exposure were found to be presence of smoker, number of smokers, identity of smoker, number of cigarettes smoked in the household and child age with the youngest children suffering twice the exposure of older children. Non-determinants were exposure to smoke other than from the parent, sex of the child, season of the year and several socioeconomic factors including civil and employment status of the mother, mother's age and educational background and whether food stamps were being received. Results of a multiple regression analysis showed that our predictors accounted for 40% of cotinine appearing in the urine. Reasons for this relatively low value may be due in part to precision of our analytic method and lower levels of ambient smoke in our population vs. others that reported higher $R^2$ values. Predictions from questionnaire information for high ETS exposure were not always the same as those indicated by urinary cotinine emphasizing that the bioindicator, which indicates the actual inhalation of ETS, is a better predictor of exposure than responses from a questionnaire.**

## Introduction

Valuable information has been obtained about environmental tobacco smoke (ETS) exposure in children via the use of questionnaires and subsequent validation using biomarkers (usually cotinine in blood, saliva or urine; Watts, Langone, Knight, & Lewtas, 1990). The amount of exposure has been shown to be influenced by human and environmental factors and major determinants have been quantitated in several populations in the United States and in Europe (CEPA, 1997; Chilmonczyk, Knight, Palomaki, Pulkkinen, Williams, & Haddow, 1990; Cook, Whincup, Jarvis, Stachan, Papacosta, & Bryant, 1994; Coultas, Howard, Peake, Skipper, & Samet, 1987; Greenberg, Haley, Etzel, & Loda, 1984; Greenberg *et al.*, 1989, 1991; Henschen *et al.*, 1997; Irvine *et al.*, 1997; Jarvis, Strachan, & Feyerabend, 1992; Jarvis *et al.*, 1985; Marbury, Hammond, & Haley, 1993; Murray & Morrison, 1988; Oddoze *et al.*, 1999; Pirkle, Fiegal, Bernert, Brody, Etzel, & Maurer, 1996; Ronchetti *et al.*, 1994; Scherer, Meger-Kossien, Reidel, Renner, & Meger, 1999). However, risk factors established for these populations are not necessarily transferable to populations in other areas of the world.

Recently, we reported findings of a pilot study of ETS exposure in a population of children (aged 2–12 years)

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residing in a tropical environment (Preston, Ramos, Calderon, & Sahai, 1997). In it, we identified children aged 2–4 years to have high risk of exposure, especially to smoke from their parents; however, small sample size precluded statistical treatment of socioeconomic and seasonal information, which when analyzed have shown to be predictive of risk in other populations (Cook *et al.*, 1994; Irvine *et al.*, 1997; Jarvis *et al.*, 1992; Ronchetti *et al.*, 1994; Scherer *et al.*, 1999). We now report a complete analysis of our data identifying major determinants to exposure and compare our results with those carried out in temperate climates.

## Methods

### *Study participants*

Our study group consisted of 606 healthy children aged 2–12 years, routinely visiting the Pediatric Primary Care Clinic of the Cataño Health Center, a satellite program of the University of Puerto Rico Pediatrics Department. Cataño is an industrial city of about 42,000 inhabitants situated across the harbor from San Juan. 'Blue collar' workers make up the bulk of the population.

Cataño is highly homogeneous in socioeconomic terms and children visiting the clinic are representative of children residing in the community as a whole. However, the manner of selection, basically by convenience, is classified as a non-probability sample and results from this study should be interpreted as suggestive in nature with conclusions not being applicable to all children living in Puerto Rico.

### *Data collection*

*Smoking and socioeconomic questionnaire.* This questionnaire was administered during the period from August 1993 through November 1996. Upon entering the clinic, mothers of the children were given an institutionally approved informed consent for participation in the study. Almost everyone approached agreed to participate but failure to adhere to study protocol (inability to produce a urine sample, non-appearance for the interview, etc.) reduced the response rate to about 75%. These factors should not introduce any appreciable bias into the study since they seemed to be distributed across all sociodemographic groups. Those who agreed to participate completed a smoking questionnaire (a copy is available in English or Spanish from the first author) which was designed to identify children's exposure to ETS, to determine the source of smoke and to assess the relative importance of each. Questions were also included about the number cigarettes smoked in each household as well as exposure outside the home.

Additional questions were posed regarding socioeconomic status of the mother including civil and employment status, age, educational background and if food stamps were being received. These categories were chosen on the basis of their importance in other studies of children exposed to ETS (Brenner & Mielck, 1993; Cook *et al.*, 1994; Greenberg *et al.*, 1991; Irvine *et al.*, 1997; Jaakkola, Ruotsalainen, & Jaakkola, 1994; Jarvis *et al.*, 1985, 1992; Pirkle *et al.*, 1996).

Likewise children's age and gender were considered as determinants. In our pilot study (Preston *et al.*, 1997), as well as from work of other investigators (Cook *et al.*, 1994; Irvine *et al.*, 1997; Pirkel *et al.*, 1996), it was shown that children of younger ages (less than 4 years old) suffered a disproportionally high level of exposure as compared to older children. Consequently, three age groups were created (aged 2–4, 5–8 and 9–12 years) and comparisons made between them.

Finally, exposure to ETS was compared by season of the year using a reference period of April to September to represent 'summer' and October to March to represent 'winter'. Investigators from Northern climates have found that 'winter' exposure to ETS is significantly greater than exposure during 'summer' mainly due to differences in home-air ventilation (Murray & Morrison, 1988; Ronchetti *et al.*, 1994; Tager, Segal, Muñoz, Weiss, & Speizer, 1987).

### *Cotinine analysis*

Fasting urine samples were collected from the children and refrigerated. Cotinine concentrations were determined within 48 h using an ELISA (Solar-Care Technologies, Bethlehem, Pennsylvania). This assay is able to measure cotinine concentrations accurately down to a level of 3 ng/ml verified against gas liquid chromatography (Tappin, 1995).

To adjust for urine dilution, urinary cotinine concentrations were standardized to creatinine concentrations and expressed as cotinine:creatinine ratios (Sepovic & Haley, 1984). Creatinine was determined colorimetrically using picric acid in an alkaline environment (Cook, 1975).

### *Statistical analysis*

Average cotinine values were compared by means of analysis of variance $F$-test or Kruskal–Wallis test when appropriate (Rosner, 1995). In order to determine if the difference in cotinine concentrations between categories having at least three levels constitute a trend, a non-parametric test for ordered alternatives due to the Jonckheere and Terpstra test, was used (Daniel, 1990). To assess the magnitude of association for children's exposure to ETS from different sociodemographic and other family characteristics, frequency distributions for exposure levels were obtained for categorical variables. Pearson's $\chi^2$ test was used to describe the statistical associations between household smoke exposure and characteristics of the population (Rosner, 1995). A test for trend in ETS prevalence was performed using Armitage's test (Daniel, 1990). Determinants associated with children's smoking exposure were assessed using unadjusted odds ratios (ORs) and 95% confidence intervals (CI).

Adjusted odds ratios (ORs) were obtained using logistic regression in which each OR was adjusted for all others determinants (Hosmer & Lemeshow, 1989).

Because the distribution of urine cotinine values was highly skewed, a logarithmic transformation was applied in order to normalize the data and stabilize the variance prior to statistical analysis. For data analysis, any values below our detection limit of 3.0 ng/mg cotinine/creatinine were entered as 1.5 ng/mg (or half the detection sensitivity). Log-cotinine concentrations were regressed on determinants of exposure to estimate, after adjusting for other predictors, the relative importance of the different sources. The unadjusted geometric mean was used to describe the cotinine concentration of the study group by back-transforming the arithmetic mean of the values on the log-scale. Analysis of covariance was used to estimate the geometric mean for each level of the predictor variables adjusting for all other significant predictors. All statistical analyses were performed using the SAS software, Version 6.12 (SAS Insitute, 1989).

## Results

Previously published studies on determinants of smoke exposure in young children have reported a substantial amount of exposure due to smoke from persons other than fathers and mothers (Cook *et al.*, 1994; Coultas *et al.*, 1987; Irvine *et al.*, 1997). In our pilot study (Preston *et al.*, 1997), we found relatively little differences in urinary cotinine of children not exposed to any smoke and those exposed to smoke from persons other than father and/or mothers. Data from this study support our previous finding of no differences between non-smoke only and 'other smoke'. The tobacco-exposure questionnaire results indicated that 178 children or 29.4% of the population were not exposed to any smoke (i.e., no family or other environmental source). Urinary cotinine in these children was 3.77 ng/mg. One hundred twenty-nine children or 21.3% of the population were exposed to 'other' smoke and had a urinary cotinine of 4.20 ng/mg. Statistical comparisons between these groups showed a

**Table 1.** Frequency distribution of reported parental smoking and children's exposure to ETS from different sources ($n$ = 606)

| Parent's smoking | $n$ | Percentage |
|---|---|---|
| None | 307 | 50.7 |
| Father | 153 | 25.2 |
| Mother | 71 | 11.7 |
| Both | 75 | 12.4 |
| Any | 299 | 49.3 |
| Quantity of cigarettes | | |
| None | 307 | 50.7 |
| <½ pack per day | 149 | 24.6 |
| ½–1 pack per day | 90 | 14.9 |
| >1 pack per day | 60 | 9.8 |

**Table 2.** Characteristics of the study subjects

| Characteristic | Exposed (E) $n$ | Exposed (E) % | Not exposed (NE) $n$ | Not exposed (NE) % | Total (T) $n$ | Total (T) % | % Household exposure (E/T) | $p$ |
|---|---|---|---|---|---|---|---|---|
| Age of child (years) | | | | | | | | |
| 2–4 | 72 | 24.1 | 90 | 29.3 | 162 | 26.7 | 44.4 | |
| 5–8 | 112 | 37.5 | 119 | 38.8 | 231 | 38.1 | 48.5 | $p = 0.177$ |
| 9–12 | 115 | 38.4 | 98 | 31.9 | 213 | 35.2 | 54.0 | |
| Gender | | | | | | | | |
| Boys | 146 | 48.8 | 149 | 48.5 | 295 | 48.7 | 49.5 | |
| Girls | 153 | 51.2 | 158 | 51.5 | 311 | 51.3 | 49.2 | $p = 0.942$ |
| Mother's age (years) | | | | | | | | |
| 16–25 | 56 | 18.7 | 83 | 27.0 | 139 | 22.9 | 40.3 | |
| 26–34 | 172 | 57.5 | 149 | 48.5 | 321 | 53.0 | 53.6 | $p = 0.320$ |
| >34 | 71 | 23.8 | 75 | 24.5 | 146 | 24.1 | 48.6 | |
| Mother's civil status | | | | | | | | |
| Living with partner | 272 | 91.0 | 221 | 72.0 | 493 | 81.4 | 55.2 | |
| Living alone | 27 | 9.0 | 86 | 28.0 | 113 | 18.6 | 23.9 | $p<0.001$ |
| Food stamps | | | | | | | | |
| Receiving | 268 | 89.6 | 233 | 75.9 | 501 | 82.7 | 53.5 | |
| Not receiving | 31 | 10.4 | 74 | 24.1 | 105 | 17.3 | 29.5 | $p<0.001$ |
| Mother's education | | | | | | | | |
| 0–8 years | 62 | 20.7 | 51 | 16.6 | 113 | 18.6 | 54.9 | |
| 9–12 years | 164 | 54.8 | 178 | 58.0 | 342 | 56.4 | 48.0 | $p = 0.430$ |
| >12 years | 73 | 24.5 | 78 | 25.4 | 151 | 25.0 | 48.3 | |
| Mother's employment status | | | | | | | | |
| Employed | 37 | 12.4 | 59 | 19.2 | 96 | 15.8 | 38.5 | |
| Not employed | 262 | 87.6 | 248 | 80.8 | 510 | 84.2 | 51.4 | $p = 0.020$ |
| Season of year | | | | | | | | |
| 'Summer' | 126 | 42.1 | 147 | 47.9 | 273 | 45.0 | 46.2 | |
| 'Winter' | 173 | 57.9 | 160 | 52.1 | 333 | 55.0 | 52.0 | $p = 0.155$ |

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**Table 3.** The crude and adjusted odds ratios for children's exposure to ETS per individual determinants (without mother's age variable)

| Determinants | Crude OR | 95% Confidence interval | Adjusted OR | 95% Confidence interval |
|---|---|---|---|---|
| Child age | | | | |
| 2–4 years | 1.00 | – | 1.00 | – |
| 5–8 years | 1.18 | (0.79–1.76) | 1.05 | (0.69–1.61) |
| 8–12 years | 1.47 | (0.97–2.21) | 1.44 | (0.93–2.22) |
| Sex | | | | |
| Boy | 1.00 | – | 1.00 | – |
| Girl | 1.01 | (0.74–1.39) | 0.99 | (0.71–1.39) |
| Mother's civil status | | | | |
| Living alone | 1.00 | – | 1.00 | – |
| Living with partner | 3.92 | (2.46–6.26) | 4.20 | (2.60–6.79) |
| Food stamps | | | | |
| Not receiving food stamps | 1.00 | – | 1.00 | – |
| Food stamp recipient | 2.75 | (1.74–4.33) | 2.82 | (1.72–4.62) |
| Mother's education level | | | | |
| >8th grade | 1.00 | – | 1.00 | – |
| 0–8th grade | 0.76 | (0.50–1.15) | 1.18 | (0.76–1.82) |
| Mother's employment status | | | | |
| Employed | 1:00 | – | 1.00 | – |
| Unemployed and/or housewife | 1.68 | (1.08–2.63) | 1.15 | (0.70–1.90) |
| Season | | | | |
| Summer | 1.00 | – | 1.00 | – |
| Winter | 0.79 | (0.57–1.09) | 1.10 | (0.78–1.56) |

*t*-value of 1.08 and a *p*-value of 0.280, indicating no differences between the two groups. Hence, the category 'no exposure', used in subsequent tables, is the sum of the non-exposed children plus the 'other' exposed children with a total of 307 (50.7% of the study subjects) and should be understood to mean 'exposure to smoke from neither father and/or mother.' It should also be mentioned that the category 'father' refers to the male partner of the mother and not necessarily the biological father of the child.

Results of exposure distribution in Table 1 compare favorably to those found in other inner city, lower income populations with 50% or more of children having ETS exposure (Mannino, Siegel, Husten, Rose, & Etzel, 1996; Overpeck & Mors, 1991; USDHHS, 1986). Similarly, numbers of cigarettes smoked in our study subjects also agree closely with reports of moderate smoking behavior in Hispanics especially among Latino women (Crawford *et al.*, 1994; Marcus & Crane, 1985).

Table 2 compares the ETS exposure risk among the children classified according to age, sex and various family and socioeconomic characteristics. Data in Table 2 were further analyzed to estimate ORs per individual determinants and the results are presented in Table 3. Exposure risks were increased by a factor of more than 4 among children in households whose mothers were living with a partner (OR = 4.2, 95% CI = 2.6–6.8) and were nearly three times greater if food stamps were being received (OR = 2.8, 95% CI = 1.7–4.6). Furthermore, children from households with unemployed mothers had slightly higher prevalence of ETS than those where mothers were employed (OR = 1.2, 95% CI = 0.7–1.9). When compared to the youngest children, while not statistically significant, there is a trend indicating higher ETS in homes of older children (OR = 1.1, 95% CI = 0.7–1.6 for the middle age group and OR = 1.4, 95% CI 0.9–2.2 for the oldest children). More smoke exposure was also noted in homes where mothers have 8 years or less of schooling vs. mothers having additional years of school (OR = 1.2, 95% CI = 0.8–1.8). This inverse relationship between the level of education and amount of smoking in parents is well documented (CEPA, 1997; Chilmonczyk *et al.*, 1990; Greenberg *et al.*, 1989; Irvine *et al.*, 1997; Jaakkola *et al.*, 1994; Overpeck & Mors, 1991; Pirkle *et al.*, 1996).

The distribution of urinary cotinine concentrations for the study sample is displayed in Figure 1. The values range from non-detectable to 267 ng/ml. Four hundred thirty out of 606 children (78.6%) had detectable levels of cotinine. This finding compares favorably with the data from the Third National Health and Nutrition Examination Survey (NHANES III) that reported 87.9% of non-tobacco users had detectable levels of cotinine in their blood (Pirkle *et al.*, 1996). Likewise, cotinine levels in our study sample were higher among those reporting ETS exposure than non-exposed persons, the magnitude being greater with an increased number of exposure sources. This finding is also in agreement with the results from NHANES III which cited higher blood levels of cotinine in individuals exposed to ETS vs. those non-exposed persons (Pirkle *et al.*, 1996).

To set our cut-off level for exposure to ETS, we selected a value that would correctly classify at least 90% of persons reporting non-exposure. Hence, using 12.5 ng/mg, as a cut-off, 277 out of 307 children (91.2%)

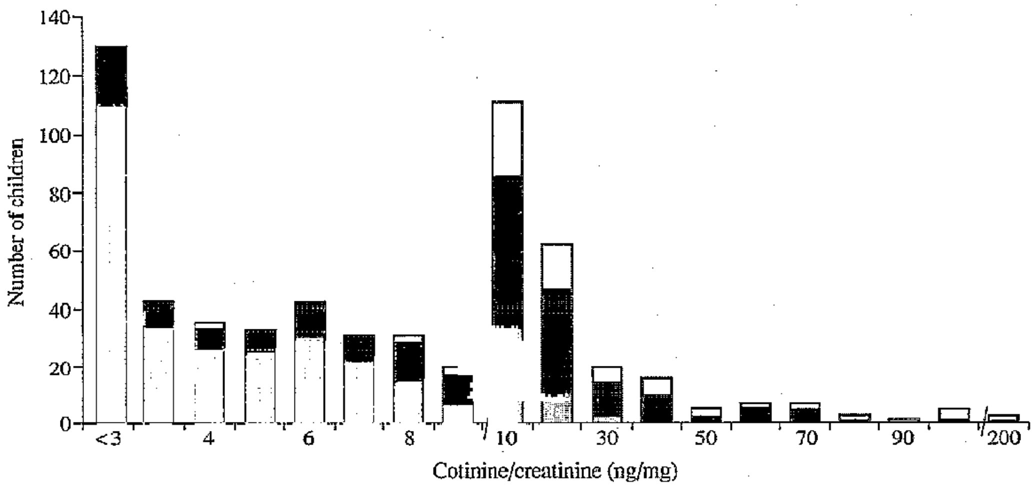


**Figure 1.** Distribution of urinary cotinine by number of sources of exposure. Dark shaded bar, non-exposed; light shaded bar, exposure to one source; open bar, exposure to two sources.

reporting non-exposure would be below this value. Similarly, 62 of 75 children (86%) whose questionnaires indicated expose to two sources would exceed this value. This cut-off is also very close to the one determined from our pilot study which was 11.6 ng/mg (Preston *et al.*, 1997).

Table 4 shows the results of the effect of sources and quantity of cigarettes on urinary cotinine. The results show a substantial increase in urinary cotinine according to exposed vs. non-exposed children, increasing number of sources of smoke, exposure to father's, mother's and combined smoke as well as exposure from an increasing number of cigarettes ($p$<0.0001). The results are comparable to those cited in literature in other studies of ETS-exposed and non-exposed children (Chilmonczyk *et al.*, 1990; Greenberg *et al.*, 1984; Scherer *et al.*, 1999; Thompson, Stone, Nanchahal, & Wald, 1990).

A comparison of multiplicative effect of the sources of exposure on urinary cotinine is shown in Table 5. It can be seen that children exposed to fathers' or mothers' smoke have an increase of nearly three times in the level of biomarker compared to non-exposed children and six times increase when children are exposed to smoke from both parents. When corrected for the number of cigarettes smoked by fathers and mothers (7.6 per day in fathers and 4.9 per day in mothers), multiplicative effects rise to 3.5 times for both fathers and mothers. Therefore smoke from either parent will result in essentially equivalent exposure levels in our population of children.

Table 6 presents the results of effects of various family, socioeconomic and seasonal factors on urinary cotinine values. Comparisons are made within each non-exposed category and within each exposed category. The use of Jonckheere–Terpstra test for trend (ordered

**Table 4.** Sources and quantity of smoking on urinary cotinine in children

| Variable | *n* | Geometric mean cotinine/creatinine (ng/mg) | 95% CI | *F*-value | *p*-value |
|---|---|---|---|---|---|
| Smoke exposure | | | | | |
| Exposed | 299 | 3.95 | (3.59–4.35) | 284.23 | <0.0001 |
| Not exposed | 307 | 14.64 | (12.99–16.50) | | |
| Number of sources | | | | | |
| None | 307 | 3.95 | (3.59–4.35) | 165.14 | <0.0001 |
| One (mother or father) | 224 | 12.28 | (10.96–14.11) | | |
| Two (mother + father) | 75 | 24.74 | (20.34–30.09) | | |
| Individual source | | | | | |
| None | 307 | 3.95 | (3.59–4.35) | 93.93 | <0.0001 |
| Father | 153 | 12.79 | (10.77–15.18) | | |
| Mother | 71 | 11.26 | (8.88–1429) | | |
| Cigarette exposure per day | | | | | |
| None | 307 | 3.95 | (3.59–4.35) | 15.78 | <0.0001 |
| <½ pack per day | 149 | 10.58 | (9.02–12.42) | | |
| ½–1 pack per day | 90 | 19.58 | (15.94–23.87) | | |
| >1 pack per day | 60 | 21.33 | (16.12–28.22) | | |

**Table 5.** Comparison of multiplicative effect of sources of exposure on urinary cotinine values

| Sources | No. | Geometric mean, ng/ml (95% CI) | Multiplicative effect compared with no exposure (95% C.I.) | Adjusted geometric mean/cotinine (ng/ml)[a] | Multiplicative effect corrected for number of cigarettes |
|---|---|---|---|---|---|
| None | 307 | 3.95 (3.59 –4.35) | 1 | – | – |
| Father | 153 | 12.79 (8.88–14.29) | 3.24 (3.01–3.47) | 13.79 (13.63–13.95) | 3.49 (3.24–3.74) |
| Mother | 71 | 11.26 (10.77–15.18) | 2.85 (2.68–3.02) | 13.74 (13.49–13.99) | 3.48 (3.32–3.64) |
| Both parents | 75 | 24.74 (20.34–30.09) | 6.26 (6.07–6.45) | – | – |

[a] Adjusted for number of cigarettes smoked per day (no adjustment made when both parents smoke).

alternatives) showed a large significant difference in cotinine levels between age groups with the youngest children exhibiting twice the value of the oldest age group ($p<0.0001$). Children of younger mothers also had higher urinary cotinine and cotinine levels were higher in children ($p = 0.005$) in households of mother living alone vs. mother living with a partner. However, both these values were quite low: 4.92 vs. 3.63, which would suggest no practical difference between the two groups. Other socioeconomic and seasonal factors had no influence on levels of the biomarker.

Table 7 gives the results of the multiple regression analysis. The results when cotinine values are regressed against the number of smoke exposure sources and other family and socioeconomic factors as predictors show that the number of smoke exposure sources, child's age and mothers years of education are of greatest importance in predicting urinary creatinine concentration. The variable 'food stamps' was found to be colinear with employment status and is consequently not included as an independent predictor. When all the predictors were entered in the equation, the percentage of the total variation in urinary cotinine, explained by the regression model, was 40%.

## Discussion

Since a high percentage of the population in the USA has measurable levels of cotinine in body fluids, it

**Table 6.** Family, socioeconomic and seasonal factors and urinary cotinine in children20expose or not exposed to ETS

| | Not exposed to ETS | | | | Exposed to ETS | | | |
|---|---|---|---|---|---|---|---|---|
| Variable | *n* | Geometric mean, ng/mg (95% CI) | *F*-value | *p*-value | *n* | Geometric mean, ng/mg (95% CI) | *F*-value | *p*-value |
| Child age | | | | | | | | |
| 2–4 years | 90 | 4.69 (3.80–5.79) | 2.60 | 0.076 | 72 | 23.37 (18.20–30.01) | 11.13 | 0.001 |
| 5–8 years | 119 | 3.69 (3.19–4.27) | | | 112 | 13.95 (11.57–16.81) | | |
| 9–12 years | 98 | 3.67 (3.15–4.56) | | | 115 | 11.45 (9.52–13.78) | | |
| Sex | | | | | | | | |
| Boys | 149 | 3.83 (3.35–4.37) | 0.38 | 0.540 | 146 | 14.13 (11.96–16.70) | 0.32 | 0.571 |
| Girls | 158 | 4.07 (3.54–4.68) | | | 153 | 15.14 (12.74–17.99) | | |
| Mother's age | | | | | | | | |
| 16–25 years | 83 | 4.55 (3.67–5.66) | 1.68 | 0.188 | 56 | 20.57 (15.31–27.63) | 3.71 | 0.026 |
| 26–34 years | 149 | 3.81 (3.35–4.34) | | | 172 | 13.40 (11.49–15.63) | | |
| >34 years | 75 | 3.61 (3.00–4.35) | | | 71 | 13.88 (10.88–17.71) | | |
| Mother's civil status | | | | | | | | |
| Living alone | 86 | 4.92 (4.11–5.88) | 7.98 | 0.005 | 27 | 11.89 (8.17–17.30) | 1.17 | 0.281 |
| Living with husband or partner | 221 | 3.63 (3.24–4.06) | | | 272 | 14.95 (13.17–16.96) | | |
| Food stamps | | | | | | | | |
| Receiving | 233 | 3.77 (3.06–4.64) | 0.30 | 0.582 | 268 | 16.67 (12.55–22.14) | 0.53 | 0.468 |
| Not receiving | 74 | 4.01 (3.60–4.47) | | | 31 | 14.42 (12.67–16.42) | | |
| Mother's education | | | | | | | | |
| 0–8th grade | 51 | 4.33 (3.47–5.40) | 3.33 | 0.037 | 62 | 15.34 (12.00–19.60) | 1.09 | 0.336 |
| 9–12th grade | 178 | 4.22 (3.72–4.80) | | | 164 | 15.43 (13.11–18.17) | | |
| >12th grade | 78 | 3.19 (2.62–3.88) | | | 73 | 12.50 (9.68–16.15) | | |
| Employment | | | | | | | | |
| Unemployed and/or housewife | 248 | 3.82 (3.44–4.25) | 1.98 | 0.160 | 262 | 14.69 (12.92–16.76) | 0.02 | 0.889 |
| Employed | 59 | 4.55 (3.60–5.74) | | | 37 | 14.31 (10.20–20.10) | | |
| Season | | | | | | | | |
| Winter | 160 | 4.06 (3.56–4.62) | 0.32 | 0.572 | 173 | 14.06 (11.99–16.49) | 0.62 | 0.433 |
| Summer | 147 | 3.84 (3.33–4.43) | | | 126 | 15.48 (12.91–18.57) | | |

**Table 7.** Factors exerting an independent significant effect on urine cotinine level of children

| Independent variable | Parameter estimates (change relative to first value) | 95% CI | Chi-square | Significance |
|---|---|---|---|---|
| Intercept | 1.96 | 1.67–2.25 | – | – |
| Smoke exposure sources | | | | |
| None | – | – | – | – |
| Smoke from one smoker parent | 1.21 | 1.05 | 221.51 | <0.001 |
| Smoke from both smoker parents | 1.94 | 1.70–2.17 | 257.85 | <0.001 |
| Child age | | | | |
| 2–4 years | – | – | – | – |
| 5–8 years | −0.32 | −0.50–(−0.13) | 10.80 | <0.001 |
| 9–12 years | −0.42 | −0.62–(−0.22) | 16.77 | <0.001 |
| Mother's age | | | | |
| 16–25 years | – | – | – | – |
| 26–34 years | −0.11 | −0.30–0.09 | 1.14 | 0.286 |
| >34 years | −0.09 | −0.31–0.14 | 0.56 | 0.455 |
| Mother's education | | | | |
| 0–8th grade | – | – | – | – |
| 9–12th grade | −0.06 | −0.26–(−0.13) | 0.42 | 0.517 |
| >12th grade | −0.30 | −0.52–(−0.07) | 6.74 | 0.009 |
| Mother's employment status | | | | |
| Employed | 0.20 | −0.01–0.40 | 3.47 | 0.062 |
| Unemployed | | | | |
| Mother's civil status | | | | |
| Living alone | −0.22 | −0.41–(−0.02) | 4.89 | 0.027 |
| Living with partner | | | | |
| Season | | | | |
| Summer | −0.05 | −0.20–0.09 | 1.48 | 0.488 |
| Winter | | | | |

$R^2 = 0.40$.

would be unrealistic to try to determine the sources for 100% of smoke exposure. A more reasonable approach would be to simply assume that a threshold of cotinine will be maintained in the body and to focus on factors that cause a major deviation from this threshold. It is important that this level of exposure be set for each population studied since unique environments will determine how smoke will be distributed. Once causative factors have been identified, that segment of the population having the highest risk potential can be pinpointed and corrective measures taken to reduce the exposure to ETS. It should be kept in mind, however, that there is no evidence of a threshold level of exposure below which it is completely safe from any disease risk (Davis, 1998).

This problem of distinguishing between ETS exposed and ETS non-exposed non-smokers has been considered by Kemmen, van Popple, Verhoef, and Jarvis (1994) who settled on a cut-off level that classifies 95% of the population within 30% of their habitual cotinine level using a single measurement. Our threshold value of 12.5 ng/mg for urinary cotinine is able to distinguish with a high degree of accuracy, persons who reported no smoke exposure vs. those reporting exposure to two source of smoke. Greater discrepancy exists in classifying persons exposed to only one source.

In regard to cut-off values, several limits have been proposed by investigators who compared cotinine in body fluids of smokers and non-smokers (Chilmonczyk *et al.*, 1990; Cummings & Richard, 1988; Etter, Duc, & Perneger, 2000; Greenberg *et al.*, 1984, 1989, 1991; Irvine *et al.*, 1997; Jarvis, Tunstall-Pedoe, Feyerabend, Vesey, & Saloojee, 1987; Jarvis *et al.*, 1985; Marbury *et al.*, 1993; Schere *et al.*, 1999; Spierto, Hannon, Kendrick, Bernert, Pirkel, & Gargiullo, 1994; Vine *et al.*, 1993). As yet, suggested cut-off values for ETS exposed populations are not available. Scherer *et al.* (1999) reported mean urinary cotinine to be 4.5 ng/mg creatinine in children residing in non-smoking households while ETS exposed children had mean urinary cotinine equal to 29.4 ng/mg. Likewise Marbury *et al.* (1993) reported values of 10 and 35 ng/mg for smoke-free children and ETS-exposed children respectively. In a study of adults either exposed or not exposed to ETS, Wall, Johnson, Jacob, and Benowitz (1988) reported cotinine/creatinine values of 6.0 and 9.2 ng/mg. These findings suggest that a cut-off value for ETS exposure should be close to our projected limit of 12.5 ng/mg especially when considering our study subjects reside in a very 'open' environment in terms of air ventilation. The important thing is not the absolute value for the quantitative amount of cotinine but that a cut-off level has been established for

minimal exposure with the understanding that values above this threshold would cause an increasingly greater risk of smoke exposure-related problems.

Although self-reported questionnaire studies of ETS have proven to provide a useful measure for determining exposure in epidemiological studies, quantification of cotinine in biological fluids is a more powerful predictor of actual exposure. This concept has been developed and refined through the work of investigators in several scientific disciplines looking at the effects of ETS and well-being of exposed individuals (Coultas *et al.*, 1987) and the superiority of bioindicators is well illustrated in the findings presented here. Data from our population characteristics (Table 2) based on questionnaire information alone show significant differences in household exposure to ETS with increased exposure levels occurring for the following characteristics: older children vs. younger children, children in households with two adults vs. mother alone and children in households receiving food stamps. When cotinine is measured, however, very different findings emerge (Table 6). Younger children rather than older children have the highest level of biomarker. There is no difference in cotinine excretion between children in households receiving or not receiving food stamps but a significant difference exists as a function of mother's education. The importance of the questionnaire data is that it provides information on the percent of the population potentially susceptible to exposure to ETS. The bioindicator actually measures the amount of smoke inhaled, hence is a truer predictor of exposure.

This same conclusion has been reacted by Perez-Stable, Benowitz, and Marin (1995) who used multivariant regression analysis models in a population of smoking women and found serum cotinine to be a better predictor of several blood components than was a questionnaire. Serum cotinine was also found to be more useful in assessing the magnitude of misclassification of smoking status in epidemiological studies than was data from a questionnaire (Riboli, Haley, Tredaniel, Saracci, Preston-Martin, & Trichopoulus, 1995).

Related to this issue, researchers should be careful to clarify apparently contradictory results such as the effect of age on ETS in our study, as the tobacco institute is adept at using mixed results as inconclusive evidence to misguide public opinion related to the risks of various tobacco-related health problems (Barnes & Bero, 1998).

On a positive note, our finding that a lower percentage of parents smoke in the presence of their youngest children is in agreement with studies elsewhere (Coultas *et al.*, 1987; Greenberg *et al.*, 1991; Preston *et al.*, 1997) where pediatricians have counseled parents about the dangers of passive smoke exposure, especially in infants. Unfortunately, even though they compose a small percentage of the population, those parents that do not heed the message cause their children to suffer an disproportionately greater risk of inhalation.

Few studies of ETS have been conducted in countries close to the equator and in those, where it was carried out, exposure was determined via questionnaire information only (De Francisco, Morris, Hall, Armstrong Schellenberg, & Greenwood, 1993; Ng, Hui, & Tan, 1993). The work reported here is the first to incorporate use of a biomarker to identify major determinants of ETS in a tropical environment. Some of the determinants we found are in common with other studies to date such as increased biomarker appearance with exposure to increased number of smokers, and increased number of cigarettes smoked by parents as well as an inverse relationship between the number of years of mother's education and number of cigarettes smoked per day (Chilmonczyk *et al.*, 1990; Cook *et al.*, 1994; Greenberg *et al.*, 1984, 1989, 1991; Irvine *et al.*, 1997; Jarvis *et al.*, 1985, 1992; Marbury *et al.*, 1993; Ronchetti *et al.*, 1994; Scherer *et al.*, 1999). Other factors such as time of year and smoke sources other than parents, which were found to be significant in other populations (Cook *et al.*, 1994; Coultas *et al.*, 1987; Irvine *et al.*, 1997; Murray & Morrison, 1988; Ronchetti *et al.*, 1994), had little influence on children in our study. Socioeconomic factors such as employment and civil status or food stamp reception showed no difference in the biomarker as a result of ETS.

Additional factors related to housing characteristics, such as the number of rooms and room size, etc., as well as air ventilation were determinants of ETS exposure in other populations (Henschen *et al.*, 1997; Irvine *et al.*, 1997; Jaakkola *et al.*, 1994; Jarvis *et al.*, 1992; Ronchetti *et al.*, 1994; Scherer *et al.*, 1999). These parameters were not considered in our study design. Such omissions may in part, account for our $R^2$ value of 0.4 which is lower than those reported by some other investigators (i.e., 0.68 for a study in children in Scotland that included home related factors (Jarvis *et al.*, 1992) and 0.72 for a study of infants in the US also testing for the effects of home size, etc. (Greenberg *et al.*, 1989) but above that of others (0.22 to 0.36 for several survey studies in German children which included home factors as determinants of ETS exposure (Henschen *et al.*, 1997).

It is also likely that part of the reason for our inability to account for a higher percentage of cotinine is that compared to active smokers and non-smokers in other populations (which reported greater $R^2$ values) urinary concentrations of cotinine in Puerto Rican children are quite low. Active smokers (>10 cigarettes per day) sustain urinary cotinine concentrations of more than 1000 ng/mg (Spierto *et al.*, 1994; Wall *et al.*, 1988) which is almost 70 times greater than our mean cotinine of 14.6 ng/mg in exposed children. Our mean value is even below that of several Northern populations exposed to ETS which have typical levels above 25 ng/mg (Spierto *et al.*, 1994).

Additionally, since our analytical method can accurately quantify cotinine down to a concentration of only 3.0 ng/ml (Sepovic & Haley, 1984), almost 20% of our baseline exposure cotinine in exposed children cannot be measured. Still, even though we are unable to account for a greater portion of cotinine in our model, those

determinants identified as being the major causes of urinary cotinine have very high statistical significance. These findings could hopefully be used to target children at highest risk and to direct information on methods to reduce this exposure in a more meaningful way.

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